IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re the Application of

Søren Mouritsen *et al.* Group Art Unit 1644

Serial No. 08/955,373 Examiner Schwadron

Filed: 21 October 1997

For: INDUCING ANTIBODY RESPONSE AGAINST SELF-PROTEINS WITH THE AID OF FOREIGN T-CELL EPITOPES

DECLARATION UNDER 37 CFR §1.132

Asst. Commissioner of Patents
Washington, D.C. 20231

Sir:

I, Dr Paul J Travers, hereby declare and state that:

1. I am Deputy Director of the Anthony Nolan Research Institute at the Royal Free Hospital in London. I hold a BA (Oxon) in the field of Biochemistry and a PhD (London) in the field of Genetics and Biometry. I have worked consistently in the field of immunology since 1979. I am *i.a.* co-author of the standard text book "Immunobiology – The Immune System in Health and Disease" published in 1999 by Current Biology Publications, part of Elsevier Science London. Other details relating to my scientific career appear from the attached Appendix A. I conclude that I am a skilled practitioner in the field of immunology. Moreover, I have since 1987 worked in the specialty of structural immunology, from 1987 to 1998 within the Department of Crystallography of Birkbeck College, London. I consider that I am knowledgable also in the field of protein structure.

2. I have read and understood the disclosure in the above-captioned US patent application, including the presently pending claims. I have further studied and understood the Office Action dated 2 May 2000 in the above-captioned US patent application. I have also studied in detail the Russell-Jones *et al.* reference cited therein.

3. I have been asked by the Applicants in the above-captioned patent application to provide my comments to a number of issues raised in the Office Action dated 2 May 2000. In the following I provide these comments.

4. In paragraph 4 in the Office Action of 2 May 2000, the Examiner states that he finds the recitation of "essentially preserve the overall tertiary structure" unclear. It is my opinion as a skilled practitioner that this is not the case.

5. "Tertiary structure of a protein" is a term generally recognized in the arts of protein chemistry and immunology and relates to the 3-dimensional structure of a protein in its normal, folded state.

6. It is my opinion that the wording "essentially preserve overall tertiary structure" as used in the above-captioned patent application can be readily understood by the skilled reader. The specification points out on page 8, line 33, that the inventive technology disclosed in the patent application results in the induction of minimal tertiary structural changes of the native protein. Further, on page 10, lines 8-13, it is indicated that exercise of the technology of the patent application minimally obscures the tertiary structure of the self-proteins. Also, in the description on page 2, it is indicated that a problem with the known technique of coupling to carrier proteins is shielding of B-cell epitopes. These 3 passages in my opinion clearly indicate to the skilled reader that "essential preservation of overall tertiary structure" implies that when a peptide containing a T-cell epitope is substituted into a self-protein according to the above-captioned patent application, the substitution is one which introduces a minimum of disturbance in the tertiary structure of the self-protein whereby a maximum number of B-cell epitopes are preserved when comparing to the unmodified self-protein.

7. The Examiner seems to argue in paragraph 4 that preservation of immunogenicity implies preservation of overall tertiary structure. This argumentation is in my opinion not in line with basic facts in immunology and protein chemistry. It has been repeatedly demonstrated that a short peptide can be immunogenic, *i.e.* capable of inducing antibodies cross-reacting with a larger protein of which the protein fragment is part. Short peptides of e.g. 12 amino acids do not have any tertiary structure.

8. Therefore, "preservation of immunogenicity" is not the equivalent of "essential preservation of overall tertiary structure". Essential preservation of overall tertiary structure does in my opinion require that a large majority of B-cell epitopes in the native protein exists in the modified protein, whereas preservation of immunogenicity can be accomplished by even one single isolated B-cell epitope from an immunogen.

9. In paragraph 6 in the Office Action of 2 May 2000, the Examiner indicates that international patent application WO 92/05192, which is hereinafter designated Russell-Jones *et al.*, describes insertion and substitution of Trat peptides into immunogens so as to preserve overall tertiary structure and argues that the preservation of immunogenicity is the equivalent of preservation of tertiary structure. As detailed above in items 7 and 8, preservation of immunogenicity is not the equivalent of preservation of overall tertiary structure.

10. The Examiner also states in paragraph 6 of the Office Action that Example 5 in Russell-Jones *et al.* provides a teaching that would be applicable to any antigen. I do, as a person skilled in immunology, not agree with this interpretation of Example 5.

11. It is in my opinion clear that Example 5 exclusively relates to a substitution strategy which will and can be used in proteins containing suppressor regions. To the best of my knowledge, there are no mammalian self-proteins which contain such suppressor regions.

12. The specific vaccine construct suggested in Example 5 of Russell-Jones *et al.* has to the best of my knowledge never been report d tested in practice. Example 5 provides no demonstration that the suggested vaccine is effective in raising antibodies against gp120, *i.e.* the immunogenicity of the suggested construct is not verified. Absent any such evidence I would, as a skilled practitioner, not be inclined to use the suggested strategy on any other protein, let alone consider that the strategy would be useful in a protein lacking suppressor regions.

13. It is on the basis of the disclosure in Russell-Jones *et al.* impossible to determine whether the suggested vaccine construct in Example 5 1) will be immunogenic and give rise to antibodies against gp120 or 2) will preserve overall tertiary structure of gp120. The example provides no considerations which point to preservation of tertiary structure, but merely suggests that a suppressor region should be replaced with a Trat epitope.

14. Apart from Example 5, I can find no suggestions in Russell-Jones *et al.* to use a vaccine preparation strategy which involves substitution in any protein.

15. The Examiner also states that the gp120 variant of Example 5 "...would be no different the claimed invention..." even if suppressor regions had no effect on immunogenicity. As a person actively working in this field of technology, I cannot agree to this line of argumentation. Example 5 in Russell-Jones *et al.* suggests a specific strategy which is devised for proteins including suppressor regions and no suggestions are given to use the strategy in proteins without suppressor regions. The strategy in Example 5 relies on substitution of the suppressor region with an immunogenic Trat peptide. In contrast, the strategy of the above-captioned patent application relies on substitution of a region so as to preserve of tertiary structure. These two strategies do not seem to be able to result in the provision of identical vaccine constructs.

16. If relying on Example 5 in Russell-Jones *et al.* to prepare a vaccine against a self-protein, Example 5 would require that the skilled reader substitutes a suppressor region in the self-protein with a Trat epitope. This would not be possible, since self-proteins are not known to include suppressor regions. Example 5 further does not provide the skilled reader with any indication to make the insertion in a self-protein so as to preserve the tertiary structure.

17. In paragraph 6 of the Office Action, the Examiner implies that LH, somatostatin, inhibin, and FSH are disclosed in Russell-Jones *et al.* for use in a vaccine to be administered to the animal where these 4 molecules are autologous. I can find no indication in Russell-Jones *et al.* which states that LH, somatostatin, inhibin, or FSH should be used for immunization in an animal where these molecules are autologous. Russell-Jones *et al.* merely states on page 9, that vaccination with these 4 molecules is contemplated, but he does not indicate anywhere that the origin of the 4 molecules is the animal species to be vaccinated.

18. Russell-Jones *et al.* refers to LH, somatostatin, inhibin, and FSH as "immunogens". In the field of immunology, one would not usually describe a self-protein as an immunogen in the autologous host. In fact, the term "immunogen" is broadly defined as a "substance" which is capable of

inducing an immune response, and in their own right that is not the case for self-proteins in the autologous host. If using a strong adjuvant together with a self-protein in order to break autotolerance towards the self-protein, I would be inclined to use the term "immunogen" for the composition of self-protein and adjuvant and not for the isolated self-protein. This is because the "substance" which is capable of inducing antibody production against the self-protein is the composition of self-protein and adjuvant, whereas none of the components in isolation have this capability.

19. Russell-Jones *et al.* provides a broad statement that "...the immunogen is any molecule it is desirable to use to raise an immune response..." The Examiner interprets this sentence as an indication that the immunogens of interest according to Russell-Jones *et al.* are any molecules it could be of interest to immunize against. However, an at least equally meaningful interpretation of this sentence would be that the immunogen could by any type of known immunogen. Further, there is no indication that the immunogens according to Russell-Jones *et al.* would include non-immunogenic self-proteins in the autologous host. Russell-Jones *et al.* merely indicates in the paragraph bridging pages 8 and 9 that the Trat peptides are useful in weak immunogens as well as in strong immunogens, but it is not indicated that the technology would be effective in breaking autotolerance towards non-immunogenic self-proteins.

20. LH, somatostatin, inhibin and FSH have all previously been reported used as immunogens in animals where they are not "self". In such cases it has been necessary to use either strong adjuvants or carrier proteins because all 4 molecules, even when derived from other species than the one to be vaccinated, are weakly immunogenic.

21. It is therefore impossible to exclude that Russell-Jones *et al.* discloses an improvement in vaccination strategies involving xenogenic LH, somatostatin, inhibin, or FSH, and it is further impossible to conclude that Russell-Jones *et al.* discloses vaccination strategies involving proteins which are autologous to the vaccinated animal.

22. I have also been asked to comment on the results obtained in Example 4 of the above-captioned patent application.

23. From Example 4 and Fig. 5 in the above-captioned patent application it can be seen that the vaccination of mice with a murine TNF-α derived vaccine construct prepared according to the principles of the above-captioned patent application resulted in an earlier onset of detectable antibody production and a higher titre of anti TNF-α antibodies than did a traditional conjugate vaccine. Therefore Example 4 demonstrates that the technology disclosed in the above-captioned patent application provides superior results compared to known technology.

24. As a skilled practitioner in the field of immunology, I find the results reported in Example 4 and Fig. 5 surprising, since neither the earlier onset of the immune response nor the higher titre of the antibodies could be predicted on the basis of the knowledge available at the time of the invention.

25. I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to

be true and further that these stat ments are made with the knowledge that wilful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such wilful false statements may jeopardize the validity of the application or any patent issuing thereon.

6 - Oct - 2000
Date

Paul Travers
Paul Travers

CURRICULUM VITAE

PERSONAL INFORMATION

Name: Travers, Paul Joseph

Born: 27 October 1956 Glasgow Scotland

Nationality: British

EDUCATION AND EXPERIENCE

1975-79 St Catherine's College, Oxford
BA (Hons) in Biochemistry (II.1)

Treasurer/Vice-President St Catherine's College Junior Common Room 1976-77
Committee member OU Scientific Society 1977-78
Committee member OU Biochemical Society 1977-78
President OU Biochemical Society 1978-79

1979-83 Imperial Cancer Research Fund, London *and*
Dept of Genetics and Biometry, University College, London

PhD supervisors Dr W F Bodmer and Prof E B Robson
PhD awarded 1984 for thesis on "The expression of HLA-ABC and other cell surface antigens by the trophoblast and tumour derived cell lines."

1984 Consultant, Intelligenetics Corp., Palo Alto, Ca.

1984-87 Research Fellow
Dept of Medical Microbiology
Stanford University Medical School

Cancer Research Institute Fellowship for studies on "The structure and evolution of the MHC", with Prof H O McDevitt.

1987-98 Research Fellow
Dept of Crystallography
Birkbeck College, University of London

Royal Society 1983 University Research Fellowship for studies on "The structure and function of MHC Class II antigens".

1999- Deputy Director, The Anthony Nolan Research Institute

FELLOWSHIPS AND AWARDS

1979 Imperial Cancer Research Fund Bursary
1984 Cancer Research Institute (NY) Fellowship
1985 NATO Travel Fellowship
1987 Royal Society 1983 University Research Fellowship
1987-1995 ICRF special project grant
1988 MRC project grant - Structure/function relationships of class II antigens
1993 SERC project grant - Isolation of cDNA and genomic clones coding for a diuretic peptide from Acheta domesticus *(with Dr K A Graeme-Cook and Dr G M Coast)*
1995 MRC project grant - Analysis of the interaction between T cell receptors and MHC:peptide complexes with agonist and antagonist peptides.
1999 Wellcome Project Grant Determination of the affinity of autoreactive TCRs and the role of CD8 *(with Dr A Cooke and Dr R Zamoyska)*

Honorary Positions and Appointments

1996 External Examiner, MSc in Biochemical and Medical Immunology, University of East London

1997 Honorary Senior Lecturer, Dept of Haematology, Royal Free Hospital School of Medicine

1998 Honorary Senior Lecturer, Dept of Crystallography, Birkbeck College

2000 Member, Subject Board in Pathology, University of London

2000 Chairman, Education Board,
British Society for Histocompatibility and Immunogenetics

PUBLICATIONS

Refereed Articles

Trowsdale J, Travers P and Bodmer WF
Expression of HLA-ABC and β_2-microglobulin antigens in human choriocarcinoma cell lines.
J Exp Med 152: 11s-17s (1980)

Adinolfi M, Ackle CA, McColl I, Fensom AH,Tansley L, Connolly P, Hsi B-L, Faulk WP, Travers P and Bodmer WF
Expression of HLA antigens, β_2-microglobulin and enzymes by human amniotic epithelial cells.
Nature 295: 325-327 (1982)

Travers PJ, Arklie JL, Trowsdale J, Patillo RA and Bodmer WF
Lack of expression of HLA-ABC antigens in choriocarcinoma and other human tumour cell lines.
National Cancer Institute Monograph 60: Research Frontiers in Ageing and Cancer NIH Publication no. 82-2436, pp175-180. (1982)

Travers PJ, Arklie JL, Trowsdale J, Patillo RA and Bodmer WF
Lack of expression of HLA-ABC antigens in choriocarcinoma and other human tumour cell lines.
Revue de l'Institut Pasteur de Lyon 16: 21-34 (1983)

Lee JS, Trowsdale J, Travers P, Carey J, Grosveld F, Jenkins J and Bodmer WF
Sequence of an HLA-DR alpha chain cDNA clone and intron-exon organisation of the corresponding gene.
Nature 299: 750-752 (1983)

Lee JS, Trowsdale J, Travers P and Bodmer WF
Molecular analysis of the HLA-D region genes
Human Immunology 8: 105-112 (1983)

Travers P and Bodmer WF
Preparation and characterisation of monoclonal antibodies against placental alkaline phosphatase and other human trophoblast associated determinants.
Int J Cancer 33: 633-641 (1984)

Epenetos AA, Travers PJ, Gatter KC, Oliver RTD, Mason DY and Bodmer WF
An immunohistochemical study of testicular germ cell tumours using two different monoclonal antibodies to placental alkaline phosphatase.
Br J Cancer 49: 11-15 (1984)

McLaughlin PJ, Travers P, McDicken IW and Johnson PM
Demonstration of placental and placental-like alkaline phosphatase in non-malignant human tissue extracts using monoclonal antibodies in an enzyme immunoassay.
Clin Chim Acta 137: 341-348 (1984)

Travers PJ, Blundell TJ, Sternberg MJE and Bodmer WF
Structural and evolutionary analysis of HLA-D region products
Nature 310: 235-238 (1984)

Trowsdale J, Lee JS, Kelly A, Jenkins J, Travers P and Bodmer WF
Isolation and sequencing of a cDNA clone for a human HLA-ABC antigen
Molecular Biology and Medicine 1: 53-61 (1984)

Trowsdale J, Lee JS, Kelly A, Carson S, Austin P and Travers P
A linkage map of two HLA-SB alpha and beta genes: an intron in one of the SB β-genes contains a processed pseudogene.
Cell 38: 241-249 (1984)

Tucker DF, Oliver RTD, Travers P and Bodmer WF
Serum marker potential of placental alkaline phosphatase-like activity in testicular

germ cell tumours evaluated by H17E2 monoclonal antibody assay
Br J Cancer 51: 631-639 (1985)

Epenetos AA, Munro AJ, Tucker DF, Gregory W, Duncan W, McDougall RH, Faux M, Travers P and Bodmer WF
Monoclonal antibody assay of serum placental alkaline phosphatase in the monitoring of testicular tumours
Br J Cancer 51: 641-644 (1985)

Grymes RA, Travers P and Engelberg A
GEL- a computer tool for DNA sequencing projects
Nucleic Acids Reseach 14: 87-98 (1986)

D J Connolly, L A Cotterill, R A Hederer, C J Thorpe, P J Travers, J H McVey, P J Dyson & P J Robinson.
A cDNA clone encoding the mouse Qa-1^a histocompatibility antigen and a proposed structure of the putative peptide binding site.
Journal of Immunology 151: 6089-6098 (1993).

Fairchild, PJ, Thorpe CJ, Travers, PJ and Wraith DC
Modulation of the immune response with T cell epitopes; the ultimate goal for specific immunotherapy of autoimmune disease
Immunology 81:487-496 (1994)

Thorpe, C.J. and Travers P.J.
Prediction of an HLA-B44 binding motif by the alignment of known epitopes and molecular modelling of the antigen binding cleft.
Immunogenetics 40: 303-305 (1994)

Thorpe, CJ, Moss, DS, Powis, SJ, Butcher, GW, Howard, JC and Travers PJ
An analysis of the antigen binding site of RT1-A^a suggests an allele specific motif
Immunogenetics 41:329-331 (1995)

Quarantino, S, Thorpe, CJ, Travers, PJ and Londei, M.
Similar antigenic surfaces rather than sequence homology dictates T cell epitope molecular mimicry.
Proc Natl Acad Sci USA 92: 10398-10402 (1995)

Gao, L, Walter, J, Travers, P, Stauss, H and Chain, BM.
The tumour associated E6 protein of human papillomavirus type 16 contains an unusual H2-K^b restricted cytotoxic T cell epitope.
Journal of Immunology 155: 5519-5526 (1995)

Amor S, O' Neill J, Morris M M, Smith R M , Wraith D C, Groome N, Travers P J and Baker D
Encephalitogenic epitopes of myelin basic protein, proteolipid protein and myelin oligodendrocyte glycoprotein for EAE induction in Biozzi ABH (H-2Ag^7) mice, share an amino acid motif.
Journal of Immunology 156:3000-3008 (1996)

Alam S M, Travers P J, Wung J L, Nasholds W, Jameson S C and Gascoigne N R J.
T cell receptor affinity and thymocyte positive selection.
Nature 381:616-620 (1996)

Jurcevic S, Travers P, Hills A, Agrewala J, Moreno C and Ivanyi J
Distinct conformations of an HLA-permissive peptide when bound to HLA-DR1 or DRB5*0101 molecules.
International Immunology 8:1807-1814 (1996)

D. McCormick, H. J. Stauss, C. J. Thorpe, P. Travers and P.J. Dyson
Major histocompatibility complex and T cell receptor interaction of the P91A tum- peptide
European Journal of Immunology 26: 2895-2902 (1996)

Lamikanra A, Gruneberg U, Altmann D, Travers_P
Mutations to the alpha-2 domain of human class II molecules alters the efficiency of peptide loading and antigen presentation
Biochemical Society Transactions, 1997, Vol.25, No.2, p.357S (1997)

Bartnes, K., Leon, F., Briand, J-P., Travers, P.J. and Hannestadt, K.
A novel first primary anchor extends the MHC class II I-A^d binding motif to encompass 9 amino acids.
International Immunology 9: 1185-1193 (1997)

Vα3.2 selection in MHC class I mutant mice: evidence for an alternate orientation of TCR-MHC class I interaction.
Sim B-C, Travers P J and Gascoigne N R J.
Journal of Immunology 159: 3323-3329 (1997)

S. Bayrak, R. Holmdahl, R. Lauster, R. Dolling, M. Hesse, P. Travers and N.A. Mitchison
T cell response of IA^q mice to self type II collagen: meshing of the binding motif of the IA^q molecule with repetitive sequence results in autoreactivity to multiple epitopes.
European Journal of Immunology 9 1687-1699 (1997)

Shidrawi, R.G., Travers, P., Rosen-Bronson, S., Wagner, A., Evan, G. and Ciclitira, P.J.
Binding of gluten peptides to HLA DQ2 (DQα1*0501,DQβ1*0201).
Clin. Exp Immunol 111: 158-165 (1998)

P. J. Robinson, P. J. Travers, A. Stackpoole, L. Flaherty and H. Djaballah
Maturation of Qa-1b class I molecules requires beta 2-microglobulin but is TAP independent
J Immunol 160: 3217-24 (1998)

M. Mata, P. J. Travers, Q. Liu, F. R. Frankel and Y. Paterson
The MHC class I-restricted immune response to HIV-gag in BALB/c mice selects a single epitope that does not have a predictable MHC-binding motif and binds to Kd through interactions between a glutamine at P3 and pocket D
J Immunol 161: 2985-93 (1998)

M. J. van Stipdonk, A. A. Willems, S. Amor, C. Persoon-Deen, P. J. Travers, C. J. Boog and J. M. van Noort
T cells discriminate between differentially phosphorylated forms of alphaB-crystallin, a major central nervous system myelin antigen
Int Immunol 10: 943-50 (1998)

S. M. Alam, G. M. Davies, C. M. Lin, T. Zal, W. Nasholds, S. C. Jameson, K. A. Hogquist, N. R. Gascoigne and P. J. Travers
Qualitative and quantitative differences in T cell receptor binding of agonist and antagonist ligands
Immunity 10: 227-37 (1999)

K. Bartnes, F. Leon, J. P. Briand, P. J. Travers and K. Hannestad
N-terminal elongation of a peptide determinant beyond the first primary anchor improves binding to H-2 I-Ad and HLA-DR1 by backbone-dependent and aromatic side chain-dependent interactions, respectively
Eur J Immunol 29: 189-95 (1999)

Bartnes, K., Li, X., Briand, J. P., Travers, P. J., and Hannestad, K. The P9 peptide sidechain specificity of I-Ad. Immunol Lett 70: 199-202. (1999)

Thoua, N., van Noort, J. M., Baker, D., Bose, A., van Sechel, A. C., van Stipdonk, M. J., Travers, P. J., and Amor, S. 2000. Encephalitogenic and immunogenic potential of the stress protein alphaB- crystallin in biozzi ABH (H-2A(g7)) mice. J Neuroimmunol 104: 47-57. (2000)

G. Fischer, M. Perez-Rodriguez, J. R. Arguello, S. T. Cox, A. McWhinnie, P. J. Travers and J. A. Madrigal
Three novel MICB alleles
Tissue Antigens 55: 166-170 (2000)

N. R. Gascoigne, S. M. Alam, C. M. Lin, M. V. McGuire, S. Marine, N. Niederberger, S. Redpath, B. C. Sim, P. J. Travers, P. Yachi, M. A. Zal & T. Zal
T cell receptor binding kinetics and special role of Valpha in T cell development and activation.
Immunol Res 21: 225-231 (2000)

Sequence-specific oligonucleotide probing for MICB alleles reveals associations with MICA and HLA-B.
G. Fischer, J. R. Arguello, M. Perez-Rodriguez, A. McWhinnie, S. G. Marsh, P.J. Travers & J. A. Madrigal
Sequence-specific oligonucleotide probing for MICB alleles reveals associations with MICA and HLA-B.
Immunogenetics 51: 591-599 (2000)

Christina Zamoyska, Lorna Kennedy, Amanda Sadler, Julia Bodmer and Paul Travers
Instability of the DRB5*0101 β chain: implications for the folding of MHC class II β chains
Journal of Immunology (Submitted for publication)

M. Perez-Rodriguez, J. R. Arguello, G. Fischer, S. T. Cox, E. Hossain, A. McWhinnie, P. J. Travers, S. G. E. Marsh and J. A. Madrigal
The extensive polymorphism of the MICA gene is the result of exon shuffling.
(submitted for publication)

Reviews

Travers P and McDevitt HO (1987)
Molecular genetics of class II (Ia) antigens
in The Antigens, vol VII
ed. M Sela, Academic Press (Florida), pp 147-211

Travers P.
One hand clapping
Nature 348: 393-394 (1990)

Zamoyska, R and Travers, P
T for II and T4, too
Current Biology 2: 38-40 (1992)

Thorpe C J and Travers P
Anchored Fore and Aft
Current Biology 2: 679-681 (1992)

Travers P.
Immunological Agnosia
Nature 363: 117-118 (1993)

Symposia and Compiled Volumes

Bodmer W, Bodmer J, Lee J, Spielman R, Travers P and Trowsdale J (1984)
Structure, evolution and polymorphism of the HLA-D region
in Immunogenetics - Its application to clinical medicine
Academic Press (Tokyo) pp 263-281

Bodmer W, Bodmer J, Lee J, So A, Spielman R, Travers P and Trowsdale J (1985)
Molecular genetics of the HLA-D region
in The Gorer Symposium : The Major Histocompatibility System
eds Sir P Medawar and T Lehner
Blackwells, Oxford pp 56-73

TL Blundell, G Elliot, SP Gardner, T Hubbard, S Islam, M Johnson, D Mantafounis, P Murray-Rust, J Overington, JE Pitts, BL Sibanda, J Singh, MJE Sternberg, MJ Sutcliffe JM Thornton and PJ Travers
Protein Engineering and Design
Phil Trans R Soc London B 324: 447-460 (1989)

Hubbard, TJP, Blundell, TL, Pitts, JE, Travers, PJ and Lockhart A.
The design synthesis and expression of CRYSTANOVA, a novel protein based on the γ-crystallin proteins.
in Protein Design in basic research, medicine and industry.
ed Morio Ikehara. Japanese Scientific Societies Press, Springer Verlag (1990)

Conformational change in MHC class II molecules
in Workshop on the Structure of the Major Histocompatibility Complex
eds Arnaiz-Villena, A and Parham P. Instituto Juan March de Estudios e Investigaciones, Madrid, 1992

Zamoyska R M and Travers P J
The function of αβ T cells and the role of the co-receptor molecules, CD4 and CD8.
in T cell receptor genes (eds E. Simpson , J. Bell & M. Owen) Oxford University Press. pp. 46-69 (1995).

Travers P J
The Major Histocompatibility Complex: Structure, Function and Polymorphism
in Handbook of Experimental Immunology (eds Herzenberg L A and Herzenberg L A) Blackwell Science (1997)

Books

Janeway, C.J and Travers, P.J
Immunobiology: the immune system in health and disease.
653 pages. Current Biology Ltd, London and Garland Publishing Inc, New York (1994 first edition; 1996 second edition; 1997 third edition, with M Walport and S Hughes; 1999 fourth edition, with M Walport and J D Capra.)